6.4.3.18.5 Bismuth-214. (CAS No. 014733-03-3; Atomic No. 83; Mol. wt. = 214 g/mol; Half-life = 19.9 minutes; Ingestion SF = 1.95E-13 risk/pCi; inhalation SF = 1.46E-11 risk/pCi; and external SF = 6.02E-06 risk/pCi). Bi-214 is an alpha- and beta-emitting member of the decay chain associated with Ra-226, which is part of the larger decay chain of naturally occurring U-238. Bismuth-214 has a physical half-life of 19.7 minutes and exists in secular equilibrium with the long-lived parent radionuclides of the decay chain. ICRP has assigned a value of 0.05 to f_1, the fractional absorption of bismuth from the gastrointestinal tract. Bismuth nitrate is assigned by ICRP to inhalation class D, and all other compounds are assigned class W. Bismuth is primarily deposited in the kidneys, with different fractions assumed to clear with biological half-lives of 0.6 and 5 days.

6.4.3.18.6 Cesium-137. (CAS No. 010045-97-3; Atomic No. 55; Mol. wt. = 137 g/mol; Half-life = 30.2 years; Ingestion SF = 3.16E-11 risk/pCi; inhalation SF = 1.91E-11 risk/pCi; and external SF = 2.09E-06 risk/yr-pCi/g). Cs-137 is a fission product produced in nuclear reactors and in nuclear weapons detonations. Cs-137 is rapidly absorbed into the bloodstream and distributes throughout the active tissues of the body. Metabolically, Cs-137 behaves as an analog of potassium. Its distribution throughout the body and energetic beta and gamma radiation from its daughter, Ba-137m, result in essentially whole-body irradiation (Amdur et al. 1991). The radioactive half-life of Cs-137 is 30 years. Its biological half-life in adults is 50 to 150 days, in children, 44 days. Cs-137 exists in secular equilibrium with Ba-137m, which is the major contributor to the dose received from a 0.662 MeV gamma ray. The critical organ for Cs-137 exposure is the whole body.

6.4.3.18.7 Europium-152. (CAS No. 014683-23-9; Atomic No. 63; Mol. Wt = 152 g/mol; Half-life = 13.6 years; Ingestion SF = 5.73E-12 risk/pCi; inhalation SF = 7.91E-11 risk/pCi; and External SF = 4.08E-06 risk/yr-pCi/g). See Section 6.4.3.19 for a general discussion of the chemical properties and toxicity of radionuclides.

6.4.3.18.8 Lead-212. (CAS No. 015092-94-1; Atomic No. 82; Mol. wt. = 212 g/mol; Half-life = 10.6 hours; Ingestion SF = 1.80E-11 risk/pCi; inhalation SF = 3.85E-11 risk/pCi; and external SF = 3.00E-07 risk/pCi). Pb-212 is a beta-emitting member of the decay chain associated with Th-228, which is of part of the larger decay chain of naturally occurring Th-232. Pb-212 has a physical half-life of 10.64 hours and exists in secular equilibrium with the long-lived parent radionuclides of the decay chain. ICRP has assigned a value of 0.2 to f_1, the fractional absorption of lead from the gastrointestinal tract. All commonly occurring compounds of lead are assigned by ICRP to inhalation class D. Lead is primarily deposited in the bone, and to a lesser extent in the liver and kidneys, with different fractions assumed to clear with biological half-lives of 12, 180, and 10,000 days.

6.4.3.18.9 Plutonium-238. (CAS No. 013981-16-3; Atomic No. 94; Mol. wt. = 238 g/mol; Half-life = 87.8 years; Ingestion SF = 2.95E-10 risk/pCi; inhalation SF = 2.74E-08 risk/pCi; and external SF = 1.94E-11 risk/pCi). Pu-238 is produced in reactors and is used in space power systems. It has been released to the environment due to burnup during atmospheric re-entry. Pu-238 has a radioactive half-life of 87.7 years, and decays by alpha emission. ICRP has assigned a value of 1E-05 to f_1, the fractional absorption of plutonium from the gastrointestinal tract, for oxides and hydroxides. Oxides and hydroxides are assigned by ICRP to inhalation class Y. All other commonly occurring compounds of plutonium are assigned by ICRP to inhalation class W, with a value of 1E-04 for f_1, the fractional absorption of plutonium from the gastrointestinal tract.

Plutonium, which is absorbed into the blood stream, is deposited mainly in the liver and bones (ATSDR 1989). Biological half-lives in liver and bone are assumed by ICRP as 40 years and 100 years, respectively. For dosimetric purposes, all isotopes of plutonium are assumed to be uniformly distributed over all bone surfaces at all times following deposition.

6.4.3.18.10 Plutonium-239 (for Pu-239/240). (CAS No. 015117-48-3; Atomic No. 94; Mol. wt. = 239 g/mol; Half-life = 24,100 years; Ingestion SF = 3.15E-10 risk/pCi; inhalation SF = 2.78E-08 risk/pCi; and external SF = 1.87E-11 risk/pCi). The main source of plutonium in the environment is from nuclear-weapons testing, with smaller contributions from accidents and space power systems burnup in the atmosphere. U.S. soil contains an estimated 5E-02 pCi/g of plutonium in the top 5 cm (4 in.).

Pu-239 has a radioactive half-life of 2.41E+04 years. Pu-239 decays by alpha emission, thus its mode of decay is accompanied by emission of x- and gamma radiation that are low energy and do not contribute significantly to radiation dose at environmental levels. ICRP has assigned a value of 1E-05 to f_1, the fractional absorption of plutonium from the gastrointestinal tract, for oxides and hydroxides. Oxides and hydroxides are assigned by ICRP to inhalation class Y. All other commonly occurring compounds of plutonium are assigned by ICRP to inhalation class W, with a value of 1E-04 for f_1, the fractional absorption of plutonium from the gastrointestinal tract.

Plutonium, which is absorbed into the blood stream, is deposited mainly in the liver and bones (ATSDR 1989). Biological half-lives in liver and bone are assumed by ICRP as 40 years and 100 years, respectively. For dosimetric purposes, all isotopes of plutonium are assumed to be uniformly distributed over all bone surfaces at all times following deposition.

6.4.3.18.11 Radium-226. (CAS No. 013982-63-3; Atomic No. 88; Mol. wt. = 226 g/mol; Half-life = 1,600 years; Ingestion SF = 2.95E-10 risk/pCi; inhalation SF = 2.72E-09 risk/pCi; and external SF = 1.31E-08 risk/pCi). Ra-226 is an alpha-emitting member of decay chain of naturally occurring U-238. Ra-226 has a physical half-life of 1622 years. ICRP has assigned a value of 0.2 to f_1, the fractional absorption of radium from the gastrointestinal tract. All radium compounds are assigned by ICRP to inhalation class W. Radium is primarily deposited in the bone, and is assumed by ICRP to be distributed throughout the volume of mineral bone.

6.4.3.18.12 Silver-108m D. (CAS No. 014391-65-2m D; Atomic No. 47; Mol. Wt = 108 g/mol; Half-life = 127 years; Ingestion SF = 6.05E-12 risk/pCi; inhalation SF = 7.02E-11 risk/pCi; and External SF = 5.62E-06 risk/yr-pCi/g). See Section 6.4.3.19 for a general discussion of the chemical properties and toxicity of radionuclides.

6.4.3.18.13 Thallium-208. (CAS No. 014913-50-9; Atomic No. 81; Mol. wt. = 208 g/mol; Half-life = 3.05 minutes; Ingestion SF = 1.75E-14 risk/pCi; inhalation SF = 1.36E-14 risk/pCi; and external SF =1.45E-05 risk/pCi). Tl-208 is a beta-emitting member of the decay chain associated with Th-228, which is of part of the larger decay chain of naturally occurring Th-232. Tl-208 has a physical half-life of 3.1 minutes and exists in secular equilibrium with the long-lived parent radionuclides of the decay chain. ICRP has assigned a value of 1.0 to f_1, the fractional absorption of thallium from the gastrointestinal tract. Oxides, hydroxides, halides, and nitrates are assigned by ICRP to inhalation class W, and all other commonly occurring compounds are assigned class D. ICRP assumes bismuth is deposited throughout all organs and tissues of the body, with a biological half-life of 10 days.

6.4.3.18.14 Uranium-234, -235, -238. (CAS No. 013966-29-5, 007440-61-1, respectively; Atomic No. 92; Mol. wt. = 234, 235, and 238 g/mol, respectively; Half-lives = 245,000 years, 704,000,000 years, and 4,470,000,000 years, respectively; Ingestion SF = 4.44E-11 risk/pCi, 4.27E-11 risk/pCi, respectively; inhalation SF =1.4E-8 risk/pCi, 1.3E-8 risk/pCi, and 1.24EE-8 risk/pCi, respectively; and external SF = 2.14E-11 risk/yr/pCi/g, 2.63E-7 risk/yr/pCi/g, and 1.5E-11 risk/yr/pCi/g, respectively). Natural uranium contains three isotopes: U-234, U-235, and U-238. The percent abundance of each isotope in natural uranium is, respectively, 0.006% and 99.27% (ATSDR 1990d). Uranium can be found in the earth's crust at an average concentration of 2 ppm. The ambient air concentration of uranium in the United States ranges from 0.3 to 0.011 fCi/m^3 (1fCi = 10^{-3} pCi). The

concentration in drinking water ranges from 0.07 to 653 pCi/L with a median value of 0.1 to 0.2 pCi/L. The average daily intake of uranium has been established to be 0.007 pCi/day from air (0.01 mg/day), 0.7 to 1 pCi/day from food (1 to 1.4 mg/day), and 0.6 to 2.0 pCi/day (0.83 to 2.78 mg/day) from drinking water.

In natural uranium, the radioactivity from U-238 accounts for about half the total radioactivity, and the radiation from U-234 and U-235 accounts for the other half. Uranium emits primarily alpha radiation that is unable to penetrate skin, but can travel short distances in the body if uranium is inhaled or ingested. Natural uranium emits very small amounts of gamma radiation that can penetrate the skin; therefore, little, if any, danger exists from this type of radiation from uranium (ATSDR 1990d). Moreover, no human or animal studies have definitively linked inhalation of or oral exposure to natural uranium to the development of cancer.

For noncancer health risks associated with uranium, exposure to natural concentrations of uranium in food, water, air, and soil does not appear to have any toxic effects. Animals that have had oral ingestion of, inhalation of, or dermal exposure to large amounts of uranium have developed damage to the kidney tubules, but other systems were not affected.

The only significant systemic health risk in humans from exposure to nonenriched uranium is potential damage to the kidneys. However, epidemiological studies have not noted an increase in deaths from urogenital or renal diseases, and intravenous studies have failed to identify significant damage to human kidneys following exposure to uranium (ATSDR 1990d). Overall, studies in animals and humans also indicate that exposure to uranium is unlikely to produce immunological or neurological effects. Although the data are conflicting, animal studies indicate that exposure to uranium may affect fetal weight and skeletal development in animals, and may possibly alter the ratio of male to female live births in areas where people have excessive exposure to uranium (ATSDR 1990d). With the exception of soluble salts, no oral or inhalation RfDs are available for uranium on IRIS or HEAST, nor has ATSDR established minimum risk levels for different environmental media (EPA 1995b; ATSDR 1990d).

ICRP has assigned a value of 0.05 to f_1, the fractional absorption of uranium from the gastrointestinal tract, for soluble fluorides and nitrates. The assigned inhalation class for fluorides and nitrates is class D. Less soluble fluorides and oxides are assigned a value of 0.05 to f_1, and an inhalation class W. Highly insoluble oxides are assigned a value of 0.002 to f_1, and an inhalation class Y.

Uranium is deposited primarily in the kidney and bones. Biological half-lives in kidney for different deposition fractions are assumed by ICRP as 6 days and 1500 days, respectively. Biological half-lives in bone for different deposition fractions are assumed by ICRP as 20 days and 5000 days, respectively. ICRP assumes that uranium-234 and 238 are uniformly distributed throughout the volume of mineral bone.

6.4.3.18.15 Zirconium-95. (CAS No. 013967-71-0; Atomic No. 40; Mol. Wt = 95 g/mol; Half-life = 0.175 years; Ingestion SF = 3.92E-12 risk/pCi; inhalation SF = 6.48E-12 risk/pCi; and External SF = 2.81E-06 risk/yr-pCi/g). See Section 6.4.3.19 for a general discussion of the chemical properties and toxicity of radionuclides.

6.5 Risk Characterization

Risk characterization involves estimating the magnitude of the potential adverse effects, summarizing the nature of the potential threats to public health, and evaluating the weight-of-evidence supporting risk estimates and the magnitude of uncertainty associated with those estimates. Specifically,

risk characterization involves combining the results of the exposure and toxicity assessments to provide quantitative estimations of risk. The risk characterization methods described in this Section are based on standard EPA guidance (EPA 1989a). An analysis of the uncertainties associated with the risk estimates calculated in this BRA is provided in Section 6.6, Uncertainty Analysis.

To characterize potential carcinogenic risks, probabilities that an individual will develop cancer over a lifetime of exposure to Site COPCs are estimated from projected intakes and chemical-specific dose-response information. To characterize potential noncarcinogenic risks, comparisons are made between estimates of intakes of Site COPCs and toxicity values. These methodologies, and results of the risk characterization for the WAG 4 retained sites and COPCs, are discussed in the sections below.

6.5.1 Generalized Approach

To quantify human health risks, contaminant intakes are calculated for each COPC by way of each applicable exposure route (see Section 6.3 and Tables D-24 through D-35). As discussed in Section 6.3, these contaminant intakes are based on measured concentration estimates at each retained release site. To determine human health risks, the contaminant specific intakes are compared to the applicable chemical-specific toxicity data discussed in Section 6.4. The following subsections discuss the equations that are used to calculate risks for each retained site.

6.5.1.1 Carcinogenic Health Effects. Cancer risks related to the Site are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the Site carcinogenic COPCs. The risk of cancer from exposure to carcinogens is estimated by using the cancer slope factor to covert chemical intake averaged over a lifetime of exposure directly to an incremental risk of an individual developing cancer. Because the cancer slope factor is often an upper 95^{th} percentile confidence limit of the probability of response based on experimental animal data, the carcinogenic risk estimate will generally be an upper-bound estimate. This means that there is reasonable confidence that the "true risk" will not exceed the cancer risk estimated and is likely to be less than predicted.

The cancer risks calculated represent excess cancer risks that may be experienced in a lifetime under a given exposure scenario. The term "excess" refers to levels above the background cancer risk. For example, national cancer statistics indicate that each person has approximately a one-in-three chance, or 333,333 chances in one million, of developing cancer during his lifetime (ACS 1986). An individual with an excess cancer risk of one in a million (denoted as either 1E-06 or 1×10^{-6}) has a total cancer risk of 333,334 in one million of developing cancer: 333,333 chances per million from background exposures, plus one chance per million from exposure to the Site.

The following calculations are used to obtain numerical estimates, (i.e., unitless probability) of lifetime cancer risks:

$$Risk = Intake \times SF \qquad (6\text{-}18)$$

where

Risk = potential lifetime cancer risk (unitless)

SF = slope factor, for chemicals (mg/kg/day)-1, or radionuclides (pCi)-1

Intake = chemical intake (mg/kg/day), or radionuclide intake (pCi).

To develop a total risk estimate for a given release site, the contaminant risks are summed for each COPC at the site.

$$Risk_T = \Sigma Risk_i \quad (6\text{-}19)$$

where

$Risk_T$ = total cancer risk, expressed as a unitless probability

$Risk_i$ = risk estimate for the *i*th contaminant.

Similarly, the risk values for each exposure route are summed to obtain the total cancer risk for each potential carcinogen.

Permissible excess cancer risks cover a range of values. The National Contingency Plan adopted an excess cancer risk range of 1E-06 to 1E-04 (i.e., one in one million to one in ten thousand) as an acceptable risk range. Consistent with this range is recent EPA guidance which states that remediation is generally not required for excess cancer risks less than 1E-04 (EPA 1991c). In addition, several past regulatory decisions indicate that in many circumstances, excess cancer risks greater than 1E-04 are permissible (Travis and Hattemer-Frey 1988).

6.5.1.2 Noncarcinogenic Effects. Health risks associated with exposure to individual noncarcinogenic compounds are evaluated by calculating hazard quotients. The potential for health effects associated with exposure to noncarcinogens is evaluated by comparing an exposure level over a specified time period (e.g., lifetime) with a reference dose derived for a similar exposure period. This ratio of toxicity is called a hazard quotient (HQ). The HQ is the ratio of the intake rate to the RfD, as follows:

$$HQ = Intake/RfD \quad (6\text{-}20)$$

where

HQ = noncancer hazard quotient (unitless)

Intake = chemical intake (mg/kg/day)

RfD = reference dose (mg/kg/day).

The hazard index (HI) is used to determine if potential noncancer effects may be of concern; it does not predict the incidence or severity of potential health effects. HIs are calculated by summing the HQs for each chemical across all exposure routes. The HI is calculated using the following equation:

$$HI = \Sigma \frac{Intake_i}{RfD_i} \quad (6\text{-}21)$$

where

HI = hazard index (unitless)

$Intake_i$= exposure level (intake) for the *i*th toxicant (mg/kg/day)

RfD_i = reference dose for the *i*th toxicant (mg/kg/day).

In the above equation, intake and RfD are expressed in the same units and represent the same exposure time period.

A HI greater than 1.0 indicates that there may be concern for potential noncancer health effects; however, it does necessarily mean that health effects will occur. As a general rule, the greater the HI is above 1.0, the greater the level of concern.

It is important to note that the level of concern does not increase linearly as the threshold level of 1.0 is approached or exceeded because RfDs do not have equal accuracy or precision and are not based on the same severity of toxic effects (i.e., the slopes of the dose-response curves in excess of the RfD can range widely depending on the substance). For this BRA, to provide conservative estimates of HIs, individual HQs will be assumed to be additive, regardless of toxic effects or mechanisms of action.

6.5.2 Estimates of Human Health Risk

Estimates of WAG 4 human health risks during each evaluated time period (see Section 6.3 for a discussion of exposure time periods) are presented in Tables D-43 through D-48 (Appendix D) and Figures 6-5 through 6-9. For each time period, carcinogenic risks and noncarcinogenic HIs are shown in separate tables and figures.

As discussed in Section 6.3, risk and HI estimates for the air and groundwater pathway exposure routes (i.e., inhalation of fugitive dust, inhalation of volatiles, ingestion of groundwater, dermal absorption of groundwater, inhalation of water vapor from indoor water use) are calculated in a cumulative manner in accordance with *Guidance Protocol for the Performance of Cumulative Risk Assessments at the INEL* (LMITCO 1995). Potential risks are estimated for COPCs in air and groundwater on a WAG-wide, instead of site-specific, basis. As a result, the estimated risk for each COPC in air and groundwater is assumed to be the same for each site retained for evaluation in the BRA. For example, the potential risk from inhalation of Cs-137 at CFA-04 is the same as the potential risk from inhalation of Cs-137 at CFA-07. Likewise, the potential future residential risk from ingestion of 1,1,1-trichloroethane at CFA-04 is the same as the potential future residential risk from ingestion of this COPC at CFA-07. This method of assessing risks for air and groundwater exposure routes is required because releases via these routes are generally not isolated and may affect all sites within the WAG. The WAG 4 air and groundwater pathway cumulative risk results are presented below in Section 6.5.2.1.

Conversely, soil exposure routes (i.e., incidental soil ingestion, dermal contact with soil, ingestion of homegrown produce) are not assessed in a cumulative manner because chemical exposures via these routes are generally isolated to a specific site (i.e., exposures are site-specific) and do not affect exposures at other sites. Site-specific risk results for WAG 4 are presented below in Section 6.5.2.2.

Risk and HQ estimates for ingestion of groundwater containing maximum predicted COPC concentrations are shown in Tables D-49 and D-50. These risk estimates are presented separately because maximum predicted COPC concentrations may occur beyond the exposure time periods evaluated in the BRA.

The following sections summarize the site-specific excess cancer and noncancer risk estimates for the current occupational worker, future occupational worker, and future resident. As discussed in

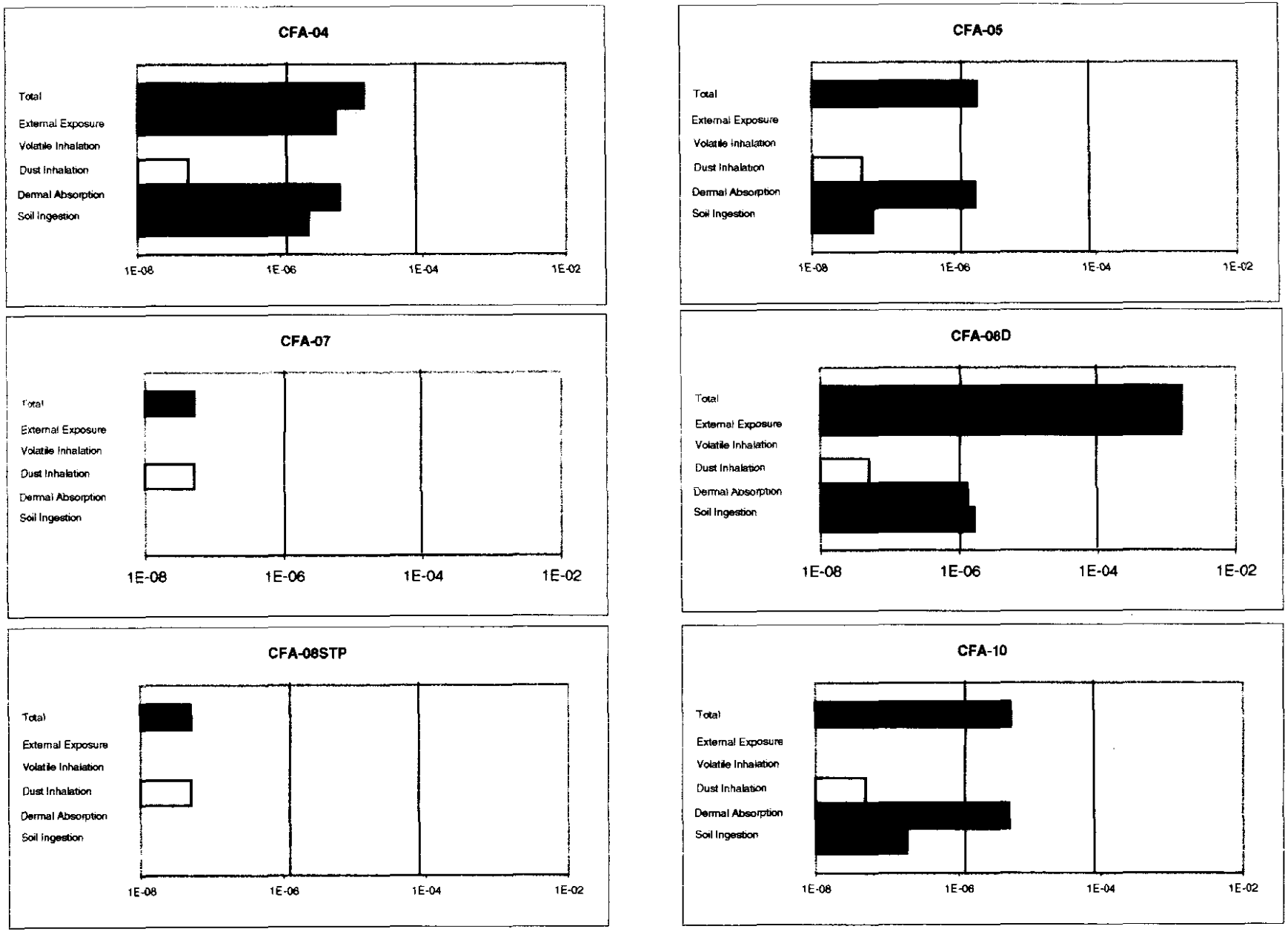


Figure 6-5. Total risk for worker at 0 years.

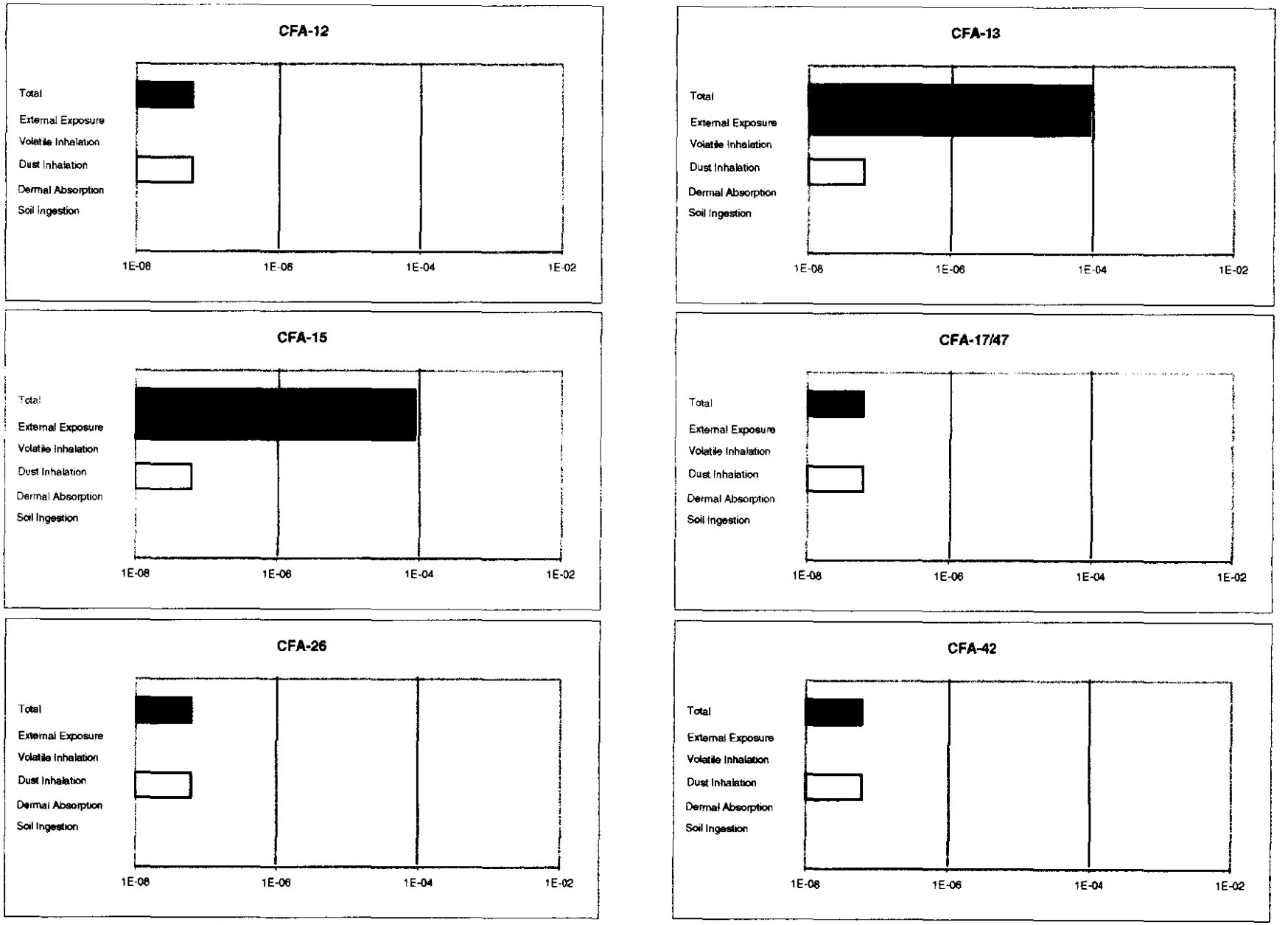
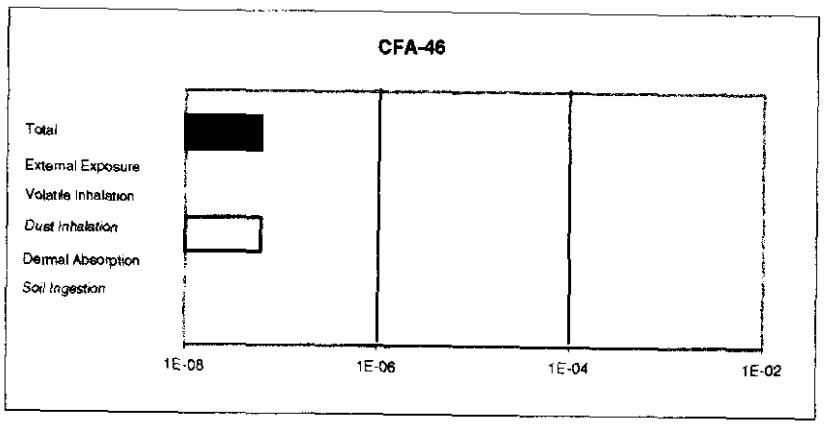


Figure 6-5. (continued).



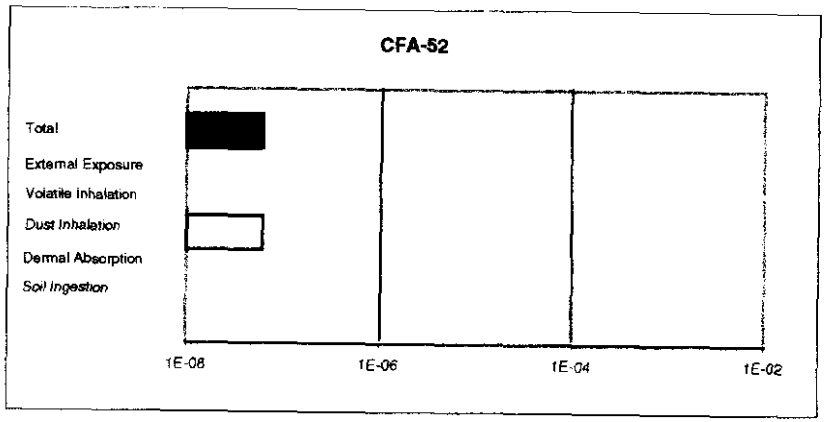


Figure 6-5. (*continued*).

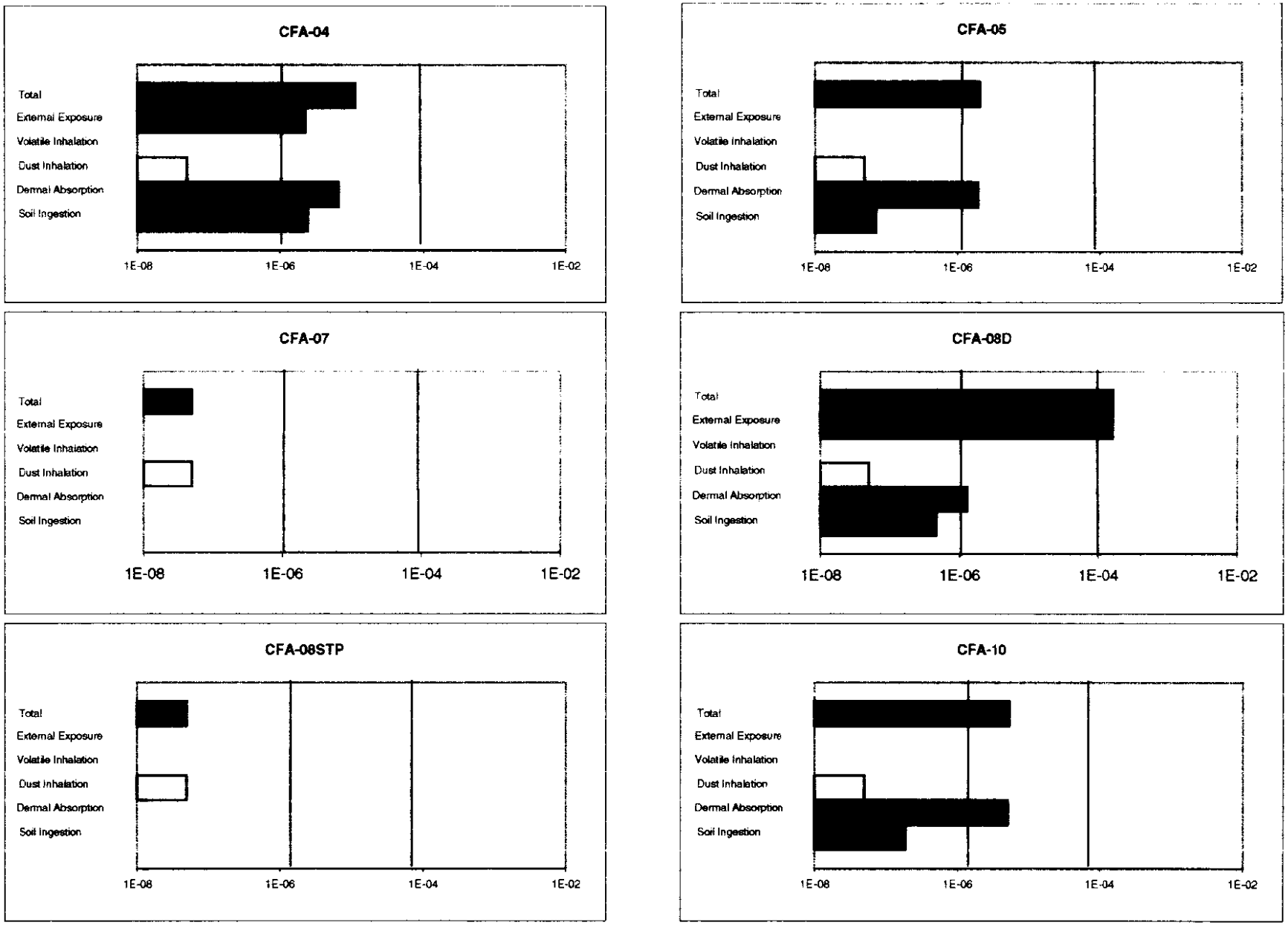
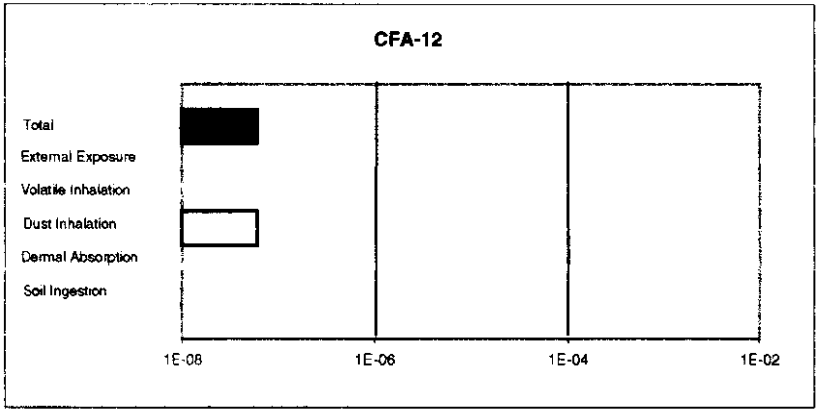
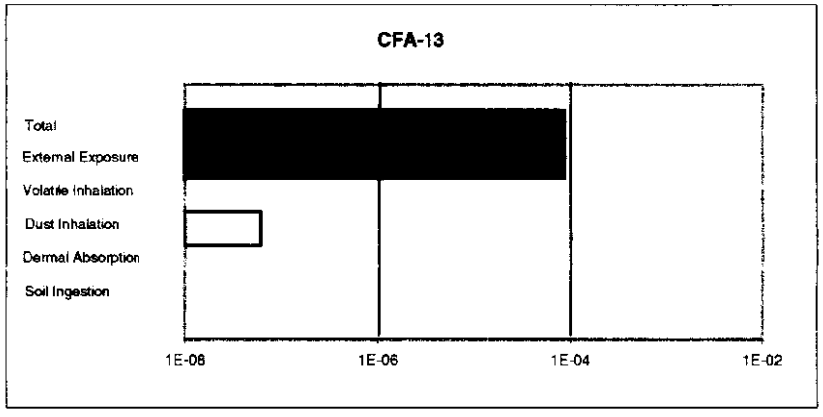
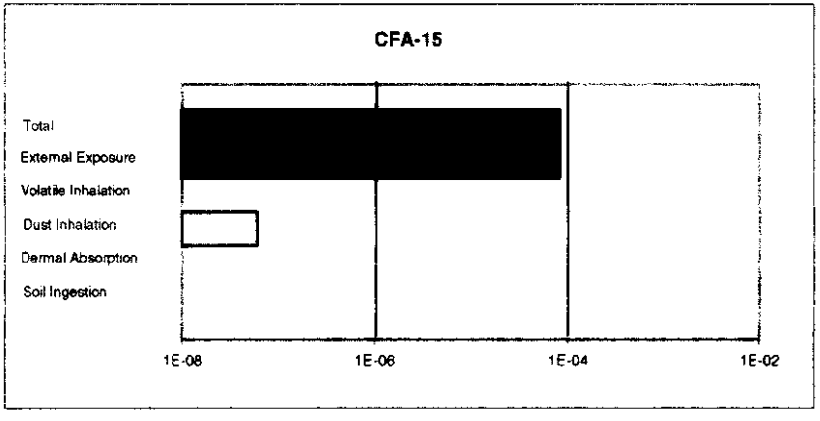
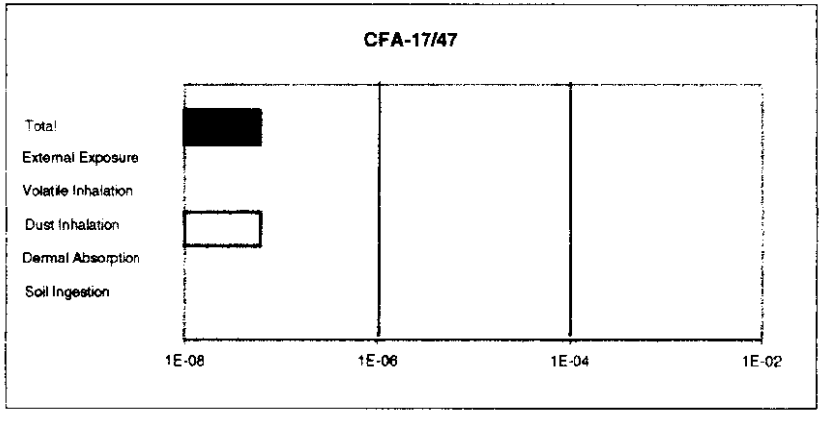


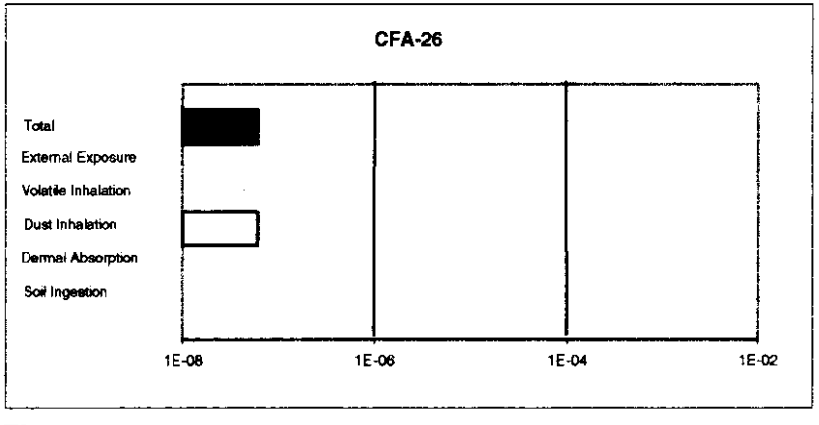
Figure 6-6. Total risk for worker at 100 years.

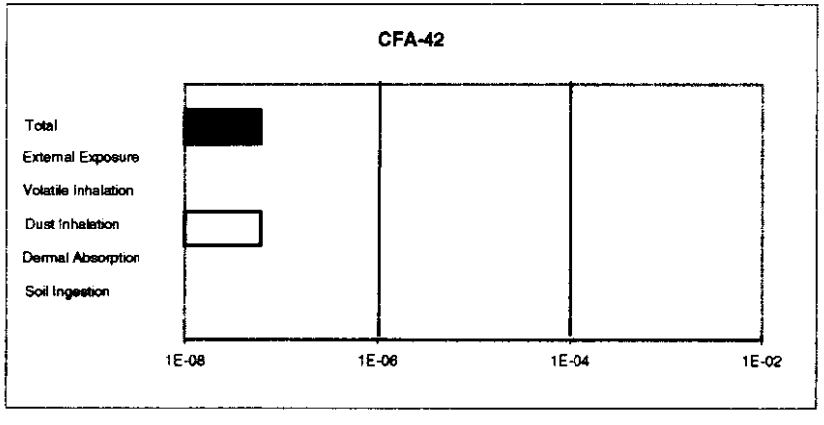












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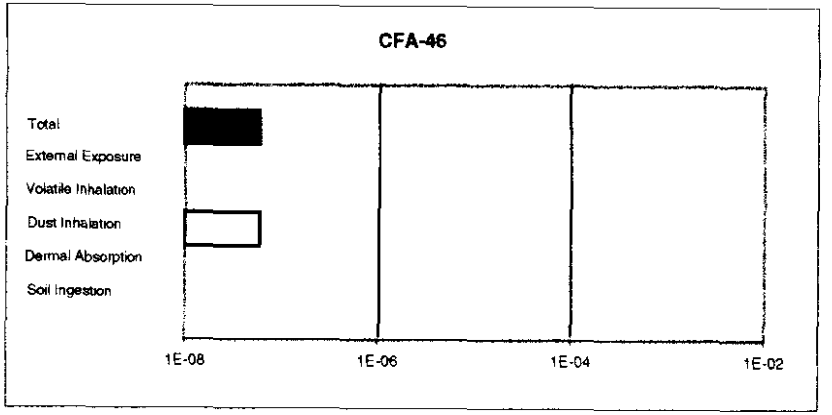

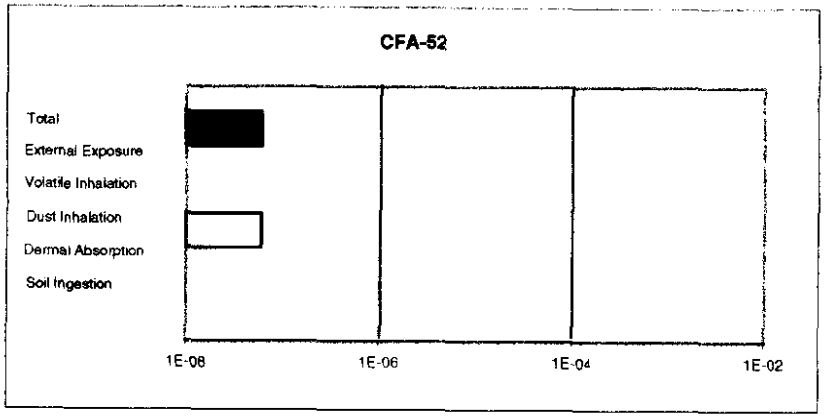

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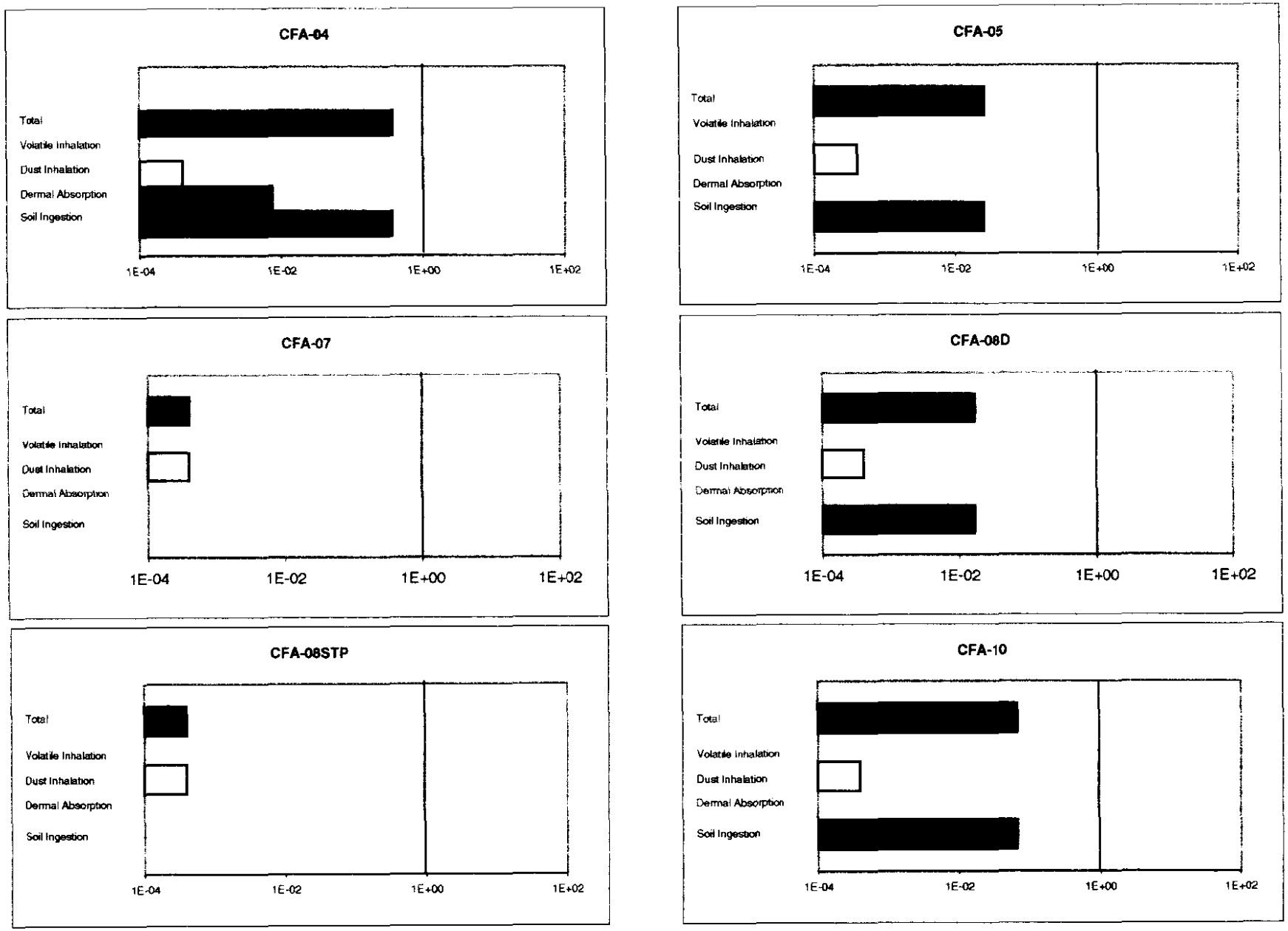
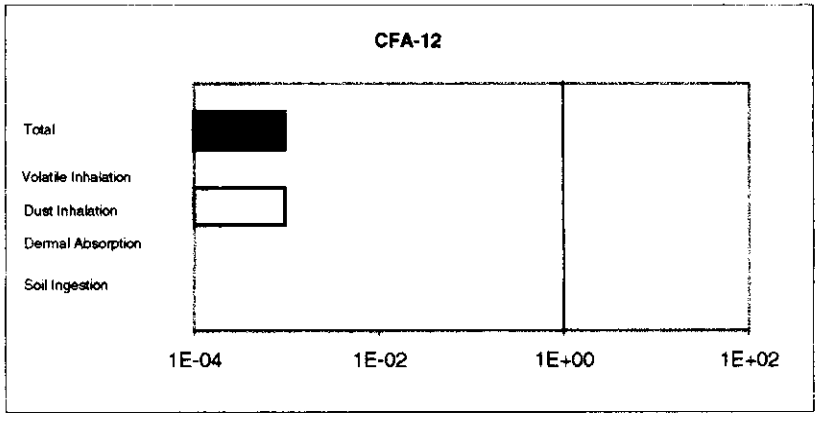
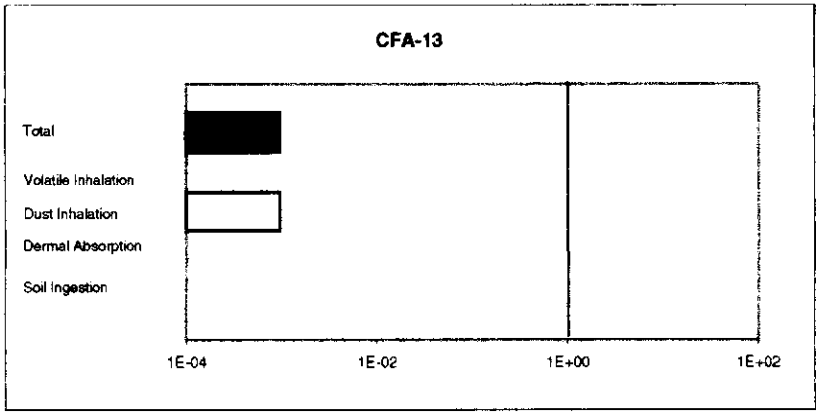
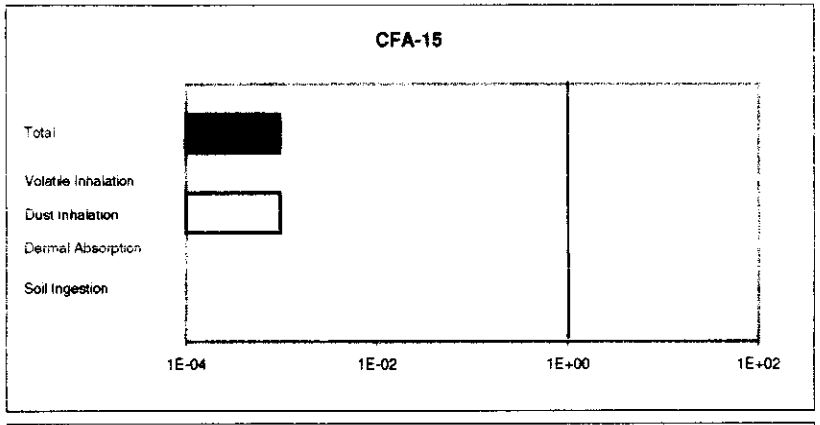
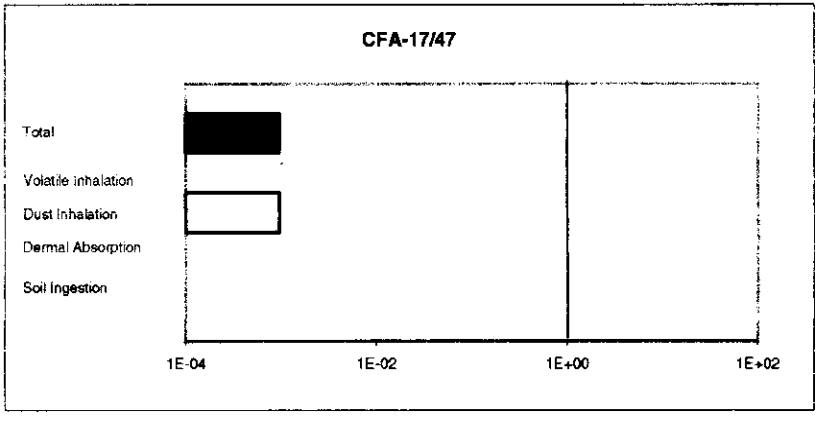


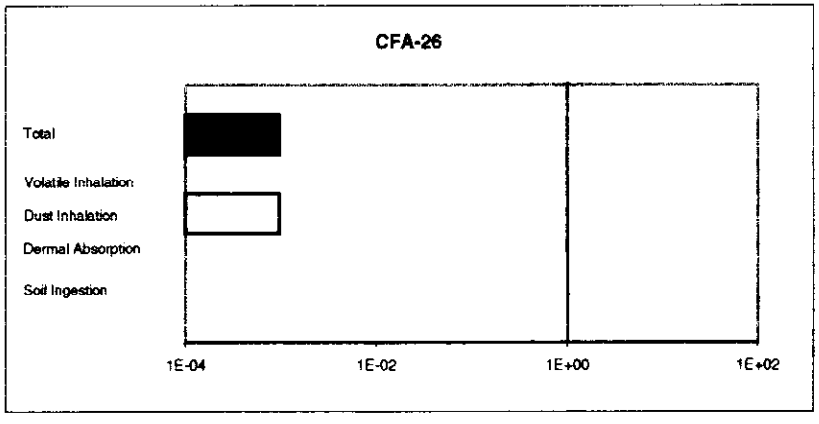
Figure 6-7. Total hazard indices for worker at 0 years.











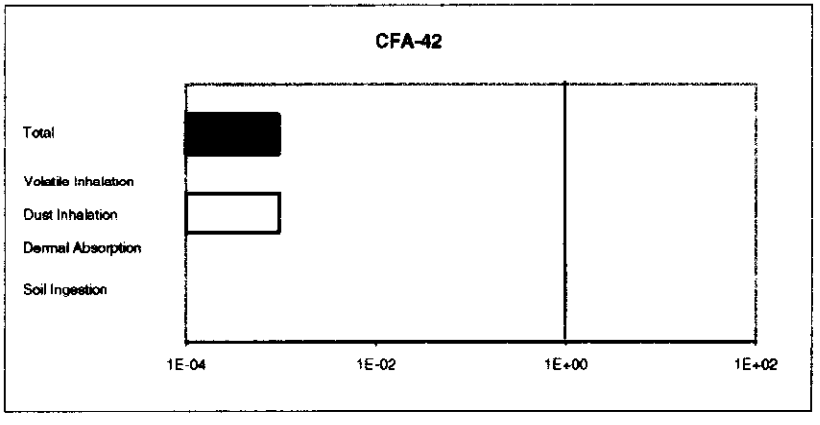
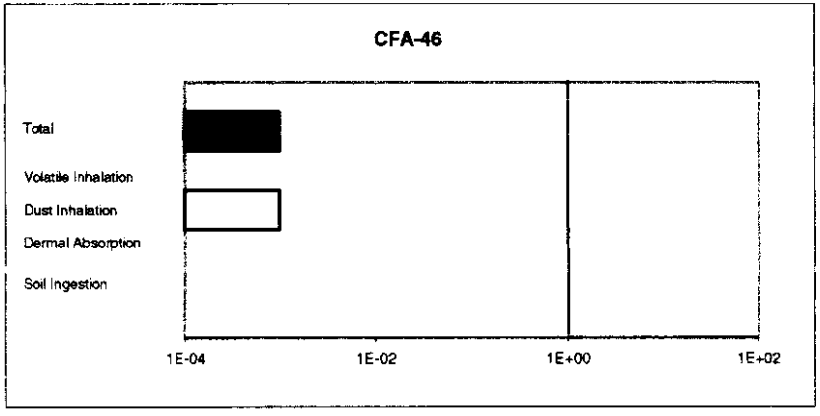


Figure 6-7. (continued).



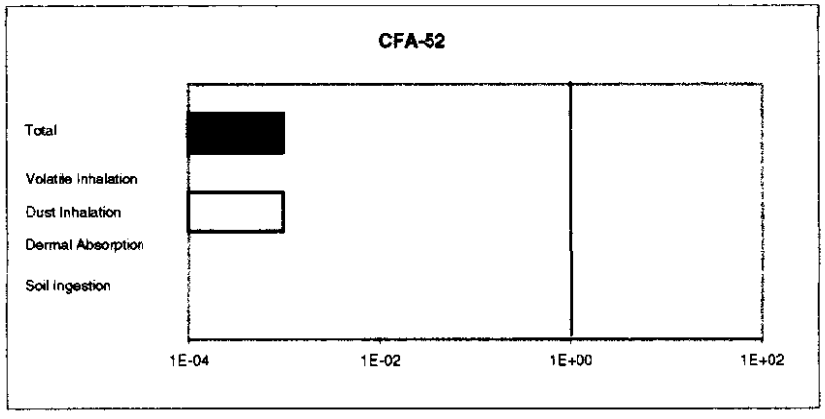


Figure 6-7. (continued).